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Search Results -

Terms	Documents
(knock-out or transgenic) near5 mouse near5 cardiotrophin-1	0

Database: US Patents Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: (knock-out or transgenic) near5 mouse near5 cardiotrophin-1 Clear

Search History

Today's Date: 12/12/2000

DB Name	Query	Hit Count	Set Name
USPT	(knock-out or transgenic) near5 mouse near5 cardiotrophin-1	0	L9
USPT	(knock-out or transgenic) near5 (animal or mouse or rat) near5 cardiotrophin-1	0	L8
USPT	cardiotropin-1 near5 gene same (knock-out or transgenic) near5 (animal or mouse or rat)	0	L7
USPT	cardiotropin-1 near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L6
USPT	(CT-1 or cardiotropin-1) near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L5
USPT	CT-1 near5 gene same (knock-out or transgenic) near5 animal or mouse or rat	84735	L4
USPT	US 5534615	1	L3
USPT	US 5627073	1	L2
USPT	US 5571893	1	L1

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Search Results -

Terms	Documents
US 5534615	1

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Refine Search: US 5534615 Clear

Search History

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DB Name	Query	Hit Count	Set Name
USPT	US 5534615	1	L3
USPT	US 5627073	1	L2
USPT	US 5571893	1	L1

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* * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 08:54:27 ON 12 DEC 2000

=> file medline, biosis, embase, caplus, scisearch

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=> s CT-1 or cardiotrophin adj 1

L1         1445 CT-1 OR CARDIOTROPHIN ADJ 1

=> s CT-1 or cardiotrophin adj 1 (p) animal or mouse or rat

   2 FILES SEARCHED...
   4 FILES SEARCHED...
L2      6973856 CT-1 OR CARDIOTROPHIN ADJ 1 (P) ANIMAL OR MOUSE OR RAT

=> s transgenic or knock adj out

L3       153725 TRANSGENIC OR KNOCK ADJ OUT

=> s L2 and L3

L4       103951 L2 AND L3

=> s cardiac hypertrophy

L5        18763 CARDIAC HYPERTROPHY

=> s L4 and L5

L6          764 L4 AND L5

=> duplicate remove L6

DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS, EMBASE, CAPLUS, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n

PROCESSING COMPLETED FOR L6
L7           325 DUPLICATE REMOVE L6 (439 DUPLICATES REMOVED)
```

=> display total ibib abs 1-5 L7

L7 ANSWER 1 OF 325 MEDLINE
ACCESSION NUMBER: 2000273924 MEDLINE
DOCUMENT NUMBER: 20273924
TITLE: Meeting Koch's postulates for calcium signaling in **cardiac hypertrophy** [comment].
COMMENT: Comment on: J Clin Invest 2000 May;105(10):1395-406
AUTHOR: Chien K R
CORPORATE SOURCE: University of California at San Diego (UCSD)-Salk Program in Molecular Medicine, UCSD School of Medicine, Basic Science Building 0613-C, 9500 Gilman Drive, La Jolla, California 92093, USA.. kchien@ucsd.edu
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (2000 May) 105 (10) 1339-42. Ref: 63
Journal code: HS7. ISSN: 0021-9738.
PUB. COUNTRY: United States
Commentary
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 200008
ENTRY WEEK: 20000803

L7 ANSWER 2 OF 325 MEDLINE
ACCESSION NUMBER: 2000273931 MEDLINE
DOCUMENT NUMBER: 20273931
TITLE: CaM kinase signaling induces **cardiac hypertrophy** and activates the MEF2 transcription factor in vivo [comment].
COMMENT: Comment on: J Clin Invest 2000 May;105(10):1339-42
AUTHOR: Passier R; Zeng H; Frey N; Naya F J; Nicol R L; McKinsey T A; Overbeek P; Richardson J A; Grant S R; Olson E N
CORPORATE SOURCE: Department of Molecular Biology, The University of Texas Southwestern Medical Center at Dallas, Dallas, Texas 75235-9148, USA.
SOURCE: JOURNAL OF CLINICAL INVESTIGATION, (2000 May) 105 (10) 1395-406.
Journal code: HS7. ISSN: 0021-9738.
PUB. COUNTRY: United States
Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 200008
ENTRY WEEK: 20000803
AB Hypertrophic growth is an adaptive response of the heart to diverse pathological stimuli and is characterized by cardiomyocyte enlargement, sarcomere assembly, and activation of a fetal program of cardiac gene expression. A variety of Ca(2+)-dependent signal transduction pathways have been implicated in **cardiac hypertrophy**, but whether these pathways are independent or interdependent and whether there is specificity among them are unclear. Previously, we showed that activation of the Ca(2+)/calmodulin-dependent protein phosphatase calcineurin or its target transcription factor NFAT3 was sufficient to evoke myocardial hypertrophy in vivo. Here, we show that activated Ca(2+)/calmodulin-dependent protein kinases-I and -IV (CaMKI and CaMKIV) also induce hypertrophic responses in cardiomyocytes in vitro and that

CaMKIV overexpressing **mice** develop **cardiac hypertrophy** with increased left ventricular end-diastolic diameter and decreased fractional shortening. Crossing this **transgenic** line with **mice** expressing a constitutively activated form of NFAT3 revealed synergy between these signaling pathways. We further show that CaMKIV activates the transcription factor MEF2 through a posttranslational mechanism in the hypertrophic heart in vivo. Activated calcineurin is a less efficient activator of MEF2-dependent transcription, suggesting that the calcineurin/NFAT and CaMK/MEF2 pathways act in parallel. These findings identify MEF2 as a downstream target for CaMK signaling in the hypertrophic heart and suggest that the CaMK and

Day : Tuesday
Date:
12/12/2000
Time:
14:06:26

PALM INTRANET

Inventor Information for 09/648183

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PENNICA,DIANE	BURLINGAME	CALIFORNIA
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WOOD,WILLIAM I.	HILLSBOROUGH	CALIFORNIA

Serial Info | Contents | Details | Atty/Agent Info | Continuity Data | Foreign Data | Inv

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